tation of empyema. Sahn and co-workers[5] reported three cases of patients with "silent" empyema on daily prednisone equivalent doses of 15, 50 and 80 mg respectively. The patient on 15 mg daily also received tetracycline.

The case described in this report differs in that neither of the two episodes was influenced by the administration of daily steroids or antibiotics prior to admission, though the patient was receiving alternate-day steroid therapy and was using antipyretic analgesic medication. Furthermore, the disease recurred after a 16-month interval and in its recurrence the pneumonia was associated with a pleural empyema.

In this case the first episode was rapidly diagnosed because the Gram stain of transtracheal aspirate suggested a bacterial cause, thus leading to prompt initiation of appropriate therapy.

In the second episode only a single symptom (pleuritic pain) was present in a patient not otherwise appearing ill. The differential diagnoses included pulmonary embolism or bacterial, fungal, viral or parasitic pneumonias. Roentgenographic features suggestive of staphylococcal pneumonia were not present, and Gram stain did not provide a clue to the cause of the illness. Transtracheal aspiration was done immediately, however, leading to a specific diagnosis in 36 hours, which was confirmed on culture of thoracic fluid. This early diagnosis obviated the need for further invasive studies. These two episodes show the potential value of transtracheal aspiration in the diagnostic approach to patients whose immune response has been compromised. This relatively simple technique, performed on admission in each instance, led to prompt diagnosis and altered subsequent management. Other invasive procedures such as bronchoscopy or open-lung biopsy procedure, often needed in such patients, were thus rendered unnecessary.

Summary

This case shows that staphylococcal pneumonia with or without empyema may have a unique, nontoxic presentation in a patient whose immune response is compromised. Transtracheal aspiration procedure may have an important role as part of the aggressive diagnostic approach to immunosuppressed patients.[6]

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Familial Hypocalciuric Hypercalcemia

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FAMILIAL BENIGN HYPERCALCEMIA was first described by Foley and his group in 1972. The term familial hypocalciuric hypercalcemia (FHH) was first used for the same disorder by Marx and coworkers in 1977. Because of its asymptomatic nature, the hypercalcemia may go undiscovered.

Few physicians are familiar with the diagnosis, and appropriate family screening is probably often not done. There may be unnecessary surgical intervention in those cases in which this condition is confused with primary hyperparathyroidism. We present a case of familial hypocalciuric hypercalcemia.

Report of a Case

A 51-year-old woman came for evaluation of persistent hypercalcemia after subtotal parathyroidectomy. A serum calcium level of 10.5 mg per dl (normal 8.5 to 10.5 mg per dl) with a serum phosphorus of 3.2 mg per dl (normal 3.0 to 4.5 mg per dl) had been noted on routine examination in 1975. In April 1977 when she had a hysterectomy, serum calcium was 11.6 mg per dl and serum phosphorus was 2.6 mg per dl. She had had a routine physical examination in Janu-

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ary 1979, at which time her serum calcium was 11.5 mg per dl. Parathyroid hormone (PTH) was reported to be 106 microliter equivalents per ml, with the normal range being 40 to 100 microliter equivalents per ml. Because her half sister had had subtotal parathyroidectomy, workup for multiple endocrine adenomatosis (MEA) was initiated. The results of urinary vanillylmandelic acid (VMA) and serum gastrin concentrations were normal. Intravenous pyelogram was reported within normal limits. At the time of that workup, she did not have symptoms from her hypercalcemia. She underwent subtotal parathyroidectomy in March 1979 at which time she was found to have four parathyroid glands and 3½ were removed. Histologic diagnosis was compatible with parathyroid hyperplasia. Serum calcium levels about a year after her surgery were elevated to 11.2 and 11.4 mg per dl. She continued to be asymptomatic.

Family history. One half sister had had symptoms of fatigue and weight loss. Her serum calcium levels ranged from 11.5 to 12.4 mg per dl. She had had subtotal parathyroidectomy (resection of 3½ parathyroid glands) in December 1975. Hyperplasia was found on microscopic examination of tissue specimen. Five years after her surgical procedure she continues to be hypercalcemic but is asymptomatic. On reviewing her records, her renal calcium to creatinine clearance ratio, based on 24-hour urine studies, was 0.007 and 0.009. Urinary calcium to creatinine clearance ratio in primary hyperparathyroidism is 0.024 ± 0.01 and in familial hypocalciuric hypercalcemia the ratio is 0.006 ± 0.004.

One half brother of the patient has been hypercalcemic for at least five years. He has refused further workup.

The father was the common parent of our patient and her half sister. He died at age 69 of arteriosclerotic disease. The autopsy report noted that two parathyroid glands were seen but were not thought to be remarkable. There was a multinodular goiter weighing about 67 grams with some calcification in its capsule. The record did not contain a serum calcium measurement. There was no notation of enlargement of the pituitary gland, and no tissue sections for histologic examination were obtained. The adrenal glands were not enlarged. There was no adenoma or hyperplasia of pancreatic islets.

One of the patient's sons has serum calcium levels towards the upper limit of normal. Another son and her daughter are normocalcemic. We were unable to discover any other first-degree relatives, either of the patient or of her father.

The patient's physical examination was noncontributory.

Laboratory studies: The patient's serum calcium levels were 11.2 and 12 mg per dl. Twenty-four-hour urinary calcium was low at 90 mg (normal 100 to 700 mg). Computerized tomography (CT) of her chest showed no mediastinal masses suggestive of an ectopic parathyroid gland. Renal calcium to creatinine clearance ratio was calculated to be 0.005. Histologic slides of parathyroid gland were reviewed by our pathologist and hyperplasia of chief cells was diagnosed.

Discussion

Familial benign hypercalcemia was first described by Foley and his group[1] in 1972. The term familial hypocalciuric hypercalcemia was first used for the same disorder by Marx and coworkers[2] in 1977. While studying families of patients with parathyroid hyperplasia, two index patients were found to belong to large kindred groups recognized as having familial hypocalciuric hypercalcemia. They did not show any features of MEA.

In FHH, hypercalcemia shows autosomal dominant transmission with almost 100 percent penetrance at all ages. Hypercalcemia begins at a very early age, that is, before age 10.[2,3]

Most of the patients do not have symptoms. Complications of renal lithiasis and peptic ulcer disease are milder and are seen less frequently than in patients with MEA or typical primary hyperparathyroidism. Twenty-four-hour urinary calcium is much lower, about a third of that seen in typical cases of primary hyperparathyroidism. Urinary calcium excretion is lower in FHH before and after adjustment for differences in creatinine clearance and serum calcium. In FHH, serum magnesium concentration is higher than in the group with primary hyperparathyroidism.[4] Ionized and ultrafiltrable plasma calcium is elevated in patients with FHH and in primary hyperparathyroidism in similar proportions to elevations in total plasma calcium. FHH patients have low urinary calcium to creatinine clearance ratio of less than 0.010 as compared with patients with primary hyperparathyroidism who have a ratio of more than 0.010.[3] Determinations of plasma PTH and cyclic adenosine monophosphate excretion are not helpful in distinguishing patients with FHH from typical primary hyperparathyroid pa-

tients. Histologically, the average amount of parathyroid parenchyma in FHH is 300 percent of that in normal parathyroid glands but considerably less than that in typical primary hyperparathyroidism.[5] Incidence of persistence or recurrence of hypercalcemia is higher in patients with FHH after parathyroidectomy than in typical primary hyperparathyroidism.

As the condition has been recognized only during the last four to five years, the best treatment for these patients remains unclear.[6] The finding of hypocalciuric hypercalcemia in several relatives favors the diagnosis of FHH. In these patients no further surgical treatment for hypercalcemia per se is indicated. The patient described in this report continues to be asymptomatic and her calcium levels have ranged from 11.5 to 12 mg per dl. No surgical intervention was recommended to her. All patients suspected of having primary hyperparathyroidism should routinely have 24-hour urinary calcium test and renal calcium to creatinine clearance ratio determination to rule out FHH. This should be done particularly if the hyperparathyroidism is diagnosed in an asymptomatic patient. If values are consistent with those seen in FHH, then family screening is in order.

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Acute Polyarthritis and Infectious Mononucleosis

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INFECTIOUS MONONUCLEOSIS (IM) is a common illness with protean manifestations ranging from subclinical infection to severe disease affecting many different organ systems. Arthralgias may occur in approximately 5 percent to 10 percent of patients.[1] Well-documented synovitis has rarely been reported. Adebonojo[2] described a 7-year-old girl with infectious mononucleosis in whom an acute inflammatory monarthritis of the ankle joint developed several days after the onset of a sore throat. This report describes an adolescent boy with severe symptoms of IM in whom an acute inflammatory polyarthritis developed.

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Report of a Case

A 16-year-old white boy developed an intensely pruritic, erythematous macular rash over his entire body. Two days later he had complaints of anorexia, fatigue, fever, severe sore throat, swelling of his hands and eyelids and photophobia. He had taken no medications before the onset of the illness. On physical examination his temperature was 38.6°C (101.5°F). There was pronounced injection of the conjunctivae, a fiery red inflammation of the oral mucosa and enlarged inflamed tonsils without exudate. There was mild cervical lymphadenopathy. The liver and spleen were not felt to be enlarged. There was a diffuse macular rash.

Initial laboratory studies showed a leukocyte count of 17,700 per cu mm with a leftward shift. There were only 4 percent lymphocytes, none of which were thought to be atypical. Wintrobe sedimentation rate was 24 mm per hour. Throat culture was negative for beta-hemolytic streptococcus and mononucleosis spot test was positive. Liver function studies were abnormal with moderate elevations in the serum aspartate aminotransferase (formerly serum glutamic oxaloacetic transaminase, SGOT) and serum alanine aminotransferase (formerly serum glutamic pyruvic transaminase, SGPT) and pronounced elevation in the alkaline phosphatase to 429 IU (30 to 100 IU). The lactic dehydrogenase (LDH) and bilirubin determinations were normal and there was no evidence of hepatitis B antigenemia. Complement-fixing antibody titers to herpes simplex,